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The Clinical Profile of Patients With Suspected Cardiogenic Shock Due to Predominant Left Ventricular Failure: A Report from the SHOCK Trial Registry

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OBJECTIVES We sought to evaluate the frequency of pulmonary congestion and associated clinical and hemodynamic findings in patients with suspected cardiogenic shock (CS).

BACKGROUND The prevalence of pulmonary congestion in the setting of CS is uncertain.

METHODS The 571 SHOCK Trial Registry patients with predominant left ventricular failure (LVF) were divided into four groups: Group A = no pulmonary congestion/no hypoperfusion = 14 (3%), Group B = isolated pulmonary congestion = 32 (6%), Group C = isolated hypoperfusion = 158 (28%) and Group D = congestion with hypoperfusion = 367 (64%). Statistical comparisons between Group C and D only, with regard to patient demographics, hemodynamics, treatment and outcome, were made.

RESULTS A significant proportion of patients with shock had no pulmonary congestion (Group C = 28%, 95% CI, 24% to 31%). Age and gender in this group were similar to Group D. Group C patients were less likely to have a prior MI ($p = 0.028$), congestive heart failure ($p = 0.005$) and renal insufficiency ($p = 0.032$), and the index MI was less likely to be anterior ($p = 0.044$). Cardiac output, cardiac index and ejection fraction were similar for the two groups but pulmonary capillary wedge pressure was slightly lower for Group C (22 vs. 24 mm Hg, $p = 0.012$). Treatment with thrombolysis, angioplasty and bypass surgery was similar in the two groups. In-hospital mortality rates for Groups C and D were 70% and 60%, respectively ($p = 0.036$). After adjustment, this difference was no longer statistically significant ($p = 0.153$).

CONCLUSIONS Absence of pulmonary congestion at initial clinical evaluation does not exclude a diagnosis of CS due to predominant LVF and is not associated with a better prognosis. (J Am Coll Cardiol 2000;36:1071–6) © 2000 by the American College of Cardiology

A critical loss of functional left ventricular (LV) myocardium in the setting of acute myocardial infarction (AMI) may result in cardiogenic shock (CS) (1–3). Cardiogenic shock is the leading cause of in-hospital death following AMI in both thrombolytic and non-thrombolytic treated populations (4). Unfortunately, patients with CS have been excluded (5) from randomized clinical trials, albeit with a few notable exceptions (6–10). Furthermore, hemodynamic confirmation has been lacking in a large proportion of study patients. As a result, clinical and hemodynamic presentation with this clinical entity remains diverse and poorly defined. The purpose of this article is to report on the prevalence of pulmonary congestion (PC) at shock onset and to correlate hemodynamic and clinical findings at shock presentation.

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The SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK? (SHOCK) Trial was a large prospective randomized study of CS complicating AMI (11). The study completed enrollment in November 1998 and the results of the randomized Trial have been published (12). Criteria for the randomized study were stringent and trial-ineligible and trial-eligible but non-consenting patients with clinical CS were prospectively enrolled in the SHOCK Trial Registry. Patients in this Registry form the subject of this report.

METHODS

Patient selection. A detailed description of the SHOCK Trial Registry methodology is reported by Hochman et al. (11,13). Registry patients with suspected CS due to predominant left ventricular failure (LVF) are the subject of these analyses. Initial case report forms did not include data for PC. Consequently, of the 884 patients with predominant LVF, 313 patients were excluded due to absence of data. In subsequent forms, these clinical findings were assessed on the basis of clinical evaluation performed at the time of initial evaluation for CS and 571 patients in the SHOCK Trial Registry had sufficient data for further

Abbreviations and Acronyms

AMI = acute myocardial infarction
BP = blood pressure
CS = cardiogenic shock
ECG = electrocardiogram, electrocardiographic
LVF = left ventricular failure
PC = pulmonary congestion
PCWP = pulmonary capillary wedge pressure
RHC = right heart catheterization
RV = right ventricular, right ventricle
SHOCK = SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK?

analysis. Four clinical groups were analyzed based on prespecified definitions for oliguria, peripheral hypoperfusion and PC (see following text): Group A = no evidence of clinical hypoperfusion or PC; Group B = PC but no evidence of clinical hypoperfusion; Group C = clinical hypoperfusion but no PC at radiographic or physical examination; and Group D = clinical hypoperfusion and PC. Of the 571 patients, 353 underwent right heart catheterization (RHC) as part of their in-hospital management. Hemodynamic data, treatment and clinical outcomes are reported for each of these subsets.

Definitions. Cardiogenic shock was defined as sustained hypotension (systolic blood pressure (BP) <90 mm Hg lasting >30 min) accompanied by evidence of tissue hypoperfusion in the setting of clinically adequate or elevated LV filling pressures. Predominant LVF was defined as the etiology of CS when none of the following shock categories was indicated: right ventricular (RV) infarction causing CS, acute severe mitral regurgitation, ventricular septal rupture, LV rupture and prior severe valvular heart disease or iatrogenic shock. Peripheral hypoperfusion was defined as the presence of cold peripheries, oliguria <30 ml/h, or both. The finding of cold peripheries was subjective and supported by physical findings of lower temperature in extremities compared with central temperature and associated cyanosis. Pulmonary congestion was defined as the presence of rales at pulmonary examination or a radiographic report of pulmonary alveolar/interstitial congestion at initial chest roentgenogram.

Statistical analysis. All continuous variables are presented as mean ± standard deviation. A two-sided p value <0.05 was considered statistically significant. Only descriptive statistics are provided for Groups A and B due to 1) their lack of a clear shock diagnosis (i.e., no hypoperfusion) and 2) their consequent small sample size. Statistical comparisons of Groups C and D only were made. The Fisher exact test was used for comparison of discrete variables, and a *t*-test or Wilcoxon test was used for continuous variables. Logistic regression was used to model the association between in-hospital mortality and clinical group (C vs. D). All patient and treatment variables that were applicable to the entire sample and had a Group C versus Group D univariate p value of ≤0.20 were evaluated for inclusion in a multivariate model for mortality. All analyses were conducted using the Statistical Analysis System (SAS for Windows, version 6.12.; SAS Institute, Cary, North Carolina).

RESULTS

Demographics. Group A consisted of 14 (3%) patients; Group B had 32 (6%); Group C had 158 patients (28%); and Group D had 367 (64%). Table 1 illustrates the profile of the patients in the four clinical groups. Although Groups C and D were comparable with respect to age and gender, patients in Group C were less likely to have had a previous MI, congestive heart failure, renal insufficiency and index anterior wall AMI. The time from AMI to onset of CS was also similar in both groups.

Table 1. Profile of Patients With Predominant LV Failure in the SHOCK Trial Registry

	Group A	Group B	Group C	Group D	p Value*
n	14	32	158	367	
Age (yrs)	59.4 ± 11.1	65.8 ± 11.1	68.8 ± 12.1	69.6 ± 11.4	0.513
Male	64.3%	53.1%	59.5%	65.1%	0.236
History of hypertension	50.0%	53.1%	51.0%	54.2%	0.555
Diabetes	7.1%	32.3%	28.5%	34.9%	0.180
History of congestive heart failure	0.0%	9.7%	12.3%	23.8%	0.005
History of renal insufficiency	0.0%	6.7%	7.1%	14.2%	0.032
Smoking	64.3%	54.8%	55.5%	54.2%	0.832
History of elevated cholesterol	38.5%	38.5%	37.8%	42.5%	0.420
History of peripheral vascular disease	0.0%	19.4%	18.6%	19.4%	0.898
History of MI	28.6%	32.3%	34.3%	45.3%	0.028
History of bypass surgery	14.3%	12.5%	15.2%	9.2%	0.061
History of angioplasty	7.1%	9.7%	6.7%	6.9%	1.000
Index anterior MI	69.2%	69.0%	49.7%	59.8%	0.044
Highest creatine kinase†	3865	2679	1904	1532	0.955
MI—shock (median hours)	15.3	10.3	4.4	6.3	0.094

*Group C vs. Group D; † = median value.
Group A = No congestion/no hypoperfusion; Group B = Isolated pulmonary congestion; Group C = Isolated hypoperfusion; Group D = Hypoperfusion with pulmonary congestion.

Table 2. Hemodynamics for Patients With Predominant LV Failure in the SHOCK Trial Registry

	Group A	Group B	Group C	Group D	p Value*
Heart rate	92.4 ± 18.2 (14)	101.8 ± 22.6 (31)	91.1 ± 27.1 (147)	96.1 ± 25.8 (351)	0.052
SBP in mm Hg	95.6 ± 13.8 (14)	102.2 ± 20.8 (31)	84.9 ± 25.5 (147)	88.0 ± 24.1 (357)	0.193
DPB in mm Hg	56.9 ± 19.4 (14)	58.3 ± 11.9 (30)	51.1 ± 19.3 (135)	51.8 ± 17.7 (311)	0.718
Lowest SBP in mm Hg	72.4 ± 11.7 (14)	51.6 ± 37.6 (32)	61.4 ± 25.6 (158)	67.5 ± 20.0 (367)	0.003
RHC	11	29	89	224	
RAP in mm Hg	13.1 ± 6.3 (8)	11.9 ± 7.1 (20)	14.6 ± 5.9 (60)	14.6 ± 8.4 (158)	0.481
PAS in mm Hg	38.4 ± 8.0 (9)	40.9 ± 14.4 (25)	38.5 ± 12.4 (80)	42.3 ± 13.0 (192)	0.008
PAD in mm Hg	23.2 ± 5.1 (9)	24.5 ± 7.9 (26)	22.3 ± 8.2 (80)	24.3 ± 8.9 (194)	0.015
PCWP in mm Hg	22.7 ± 7.8 (11)	21.9 ± 9.8 (29)	21.5 ± 6.7 (82)	24.3 ± 8.1 (209)	0.012
SVR in dynes/sec/cm^{-5}	1632 ± 853 (4)	1597 ± 579 (15)	1556 ± 702 (43)	1454 ± 693 (119)	0.538
Cardiac index in 1/min/m2	2.1 ± 0.8 (7)	2.3 ± 1.0 (21)	1.9 ± 0.6 (62)	2.1 ± 0.8 (170)	0.218
Cardiac output in 1/min	4.2 ± 2.2 (6)	4.0 ± 1.4 (20)	3.6 ± 1.3 (62)	3.9 ± 1.7 (168)	0.418
In-hospital LVEF in %	37.8 ± 16.2 (9)	27.8 ± 9.8 (20)	30.9 ± 12.5 (66)	30.4 ± 12.8 (202)	0.839

*Group C vs. Group D.

Group A = No congestion/no hypoperfusion, Group B = Isolated pulmonary congestion, Group C = Isolated hypoperfusion, and Group D = Hypoperfusion with pulmonary congestion.

( ) = number of patients with data.

SBP = systolic blood pressure, DBP = diastolic blood pressure, LVEF = left ventricular ejection fraction, PCWP = pulmonary capillary wedge pressure, RAP = right atrial pressure and RHC = right heart catheterization, SVR = systemic vascular resistance.

Values were often recorded while the patient was on hemodynamic support.

Hemodynamic data. Right heart catheterization was performed in 11 of 14 patients (79%) in Group A, in 29 of 32 (91%) in Group B, in 89 of 159 (56%) in Group C and in 224 of 367 (61%) in Group D. The hemodynamic findings at the time of RHC for suspected clinical CS are presented in Table 2. Groups C and D meet standard criteria for CS

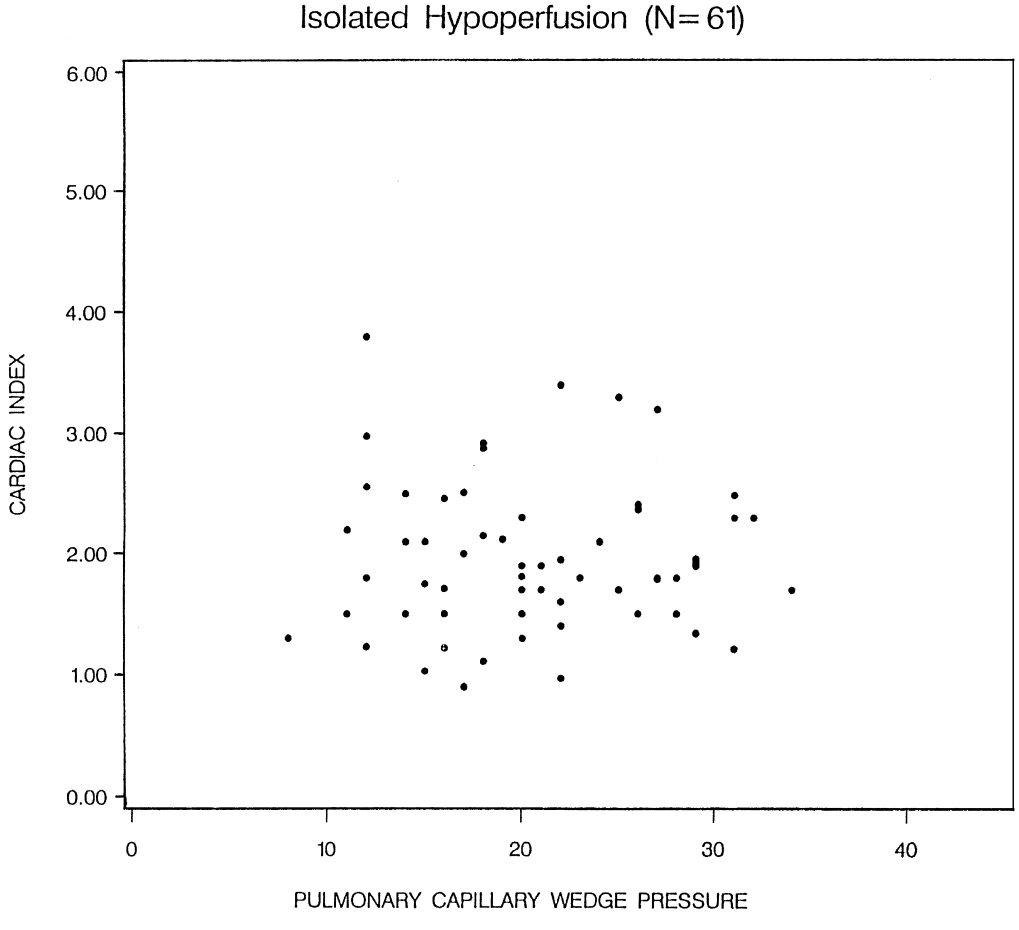

Figure 1. This figure plots PCWP against cardiac index for individual patients in Group C (isolated hypoperfusion). Because a majority of patients in this group have PCWP >15 mm Hg, occult predominant RV dysfunction in the setting of inferior infarction does not appear to play a major role in this clinical presentation.

Table 3. Treatment of Patients With Predominant LV Failure in the SHOCK Trial Registry

	Group A	Group B	Group C	Group D	p Value*
n	14	32	158	367	
Thrombolytic (%)	14.3	43.8	31.7	31.7	1.000
Coronary angiography (%)	85.7	75.0	60.8	63.5	0.557
Angioplasty (%)	64.3	37.5	35.4	30.0	0.221
Bypass surgery (%)	21.4	9.4	12.7	16.9	0.240
IABP (%)	64.3	53.1	52.5	52.9	1.000
Ventilator (%)	64.3	65.6	73.4	77.7	0.314
Intravenous vasopressor use (%)	78.6	93.8	95.6	96.7	0.611
Intravenous inotrope use (%)	57.1	75.0	60.1	76.8	< 0.001

*Group C vs. Group D.
Group A = No congestion/no hypoperfusion, Group B = Isolated pulmonary congestion, Group C = Isolated hypoperfusion, and Group D = Hypoperfusion with pulmonary congestion.

with hypotension, elevated pulmonary capillary wedge pressure (PCWP) and depressed cardiac index ≤2.2. Although the mean cardiac indexes in these groups are comparable, Group C patients had a significantly lower mean PCWP (22 ± 7 vs. 24 ± 9 mm Hg, p = 0.012) as well as lower pulmonary systolic and diastolic pressures. Other parameters, including heart rate, systolic BP, diastolic BP, right atrial pressure, systemic vascular resistance and cardiac output were comparable for Groups C and D. In-hospital LV ejection fraction was also similar in Groups C and D. Figure 1 plots PCWP against cardiac index for patients in Group C. Although this Group C has more index non-anterior MI, occult predominant RV dysfunction and volume depletion appear not to play a major role in this subset. As illustrated, a majority of patients in this group have PCWP >15 mm Hg.

Management. Almost one-third of the patients in both Groups C and D were treated with thrombolysis (Table 3). The rates of diagnostic coronary angiography, percutaneous angioplasty and bypass surgery, hemodynamic support with intra-aortic balloon pump counterpulsation and use of mechanical ventilation were comparable for Groups C and D. The overwhelming majority of patients in both groups required intravenous vasopressors as part of their medical care. However, intravenous inotropes were used less often in Group C, compared with D (60% vs. 77%, p < 0.001).

Angiography. Coronary angiography was performed on 96 patients (61%) in Group C and 233 (63%) patients in Group D. Patients in Group C had more single-vessel disease (27.5% vs. 14.6%) and less triple-vessel disease (49.5% vs. 63.5%) (p = 0.002). The distribution of left main disease in both clinical groups was comparable (16% vs. 19%, p = 0.624).

In-hospital mortality. In-hospital mortality rates for Groups A and B were 21% and 22%, respectively. The outcome of Groups C and D were poor, with in-hospital mortality of 70% and 60%, respectively (odds ratio [OR] for death 1.53, 95% confidence interval [CI] 1.03 to 2.28, p = 0.036; n = 525). After adjusting for prior MI, lowest recorded systolic BP, and inotrope administration, there was no significant difference in mortality for Groups C and D (OR for death 1.38, 95% CI 0.89 to 2.13, p = 0.153, n = 476).

DISCUSSION

In this large, prospective, international Registry of suspected CS complicating AMI we observed that the majority of patients with predominant LVF causing CS have classical findings of peripheral hypoperfusion and PC (Group D) in the setting of arterial hypotension. It is, however, remarkable that in the setting of severe acute LV dysfunction, approximately one-quarter of patients manifest hypotension and hypoperfusion in the absence of clinical PC (Group C). Unadjusted in-hospital mortality was higher in this group than that observed for patients with PC in conjunction with peripheral hypoperfusion.

The absence of PC in the presence of an elevated PCWP has been previously documented (14). Its prevalence in the setting of acute LVF complicating AMI, however, deserves emphasis. Our observations suggest that this is not an uncommon phenomenon. Consequently, the absence of PC at physical evaluation should not be considered a surrogate for low risk. Clearly, LV compromise can coexist with a normal initial pulmonary evaluation.

Pathogenesis and outcome. The absence of PC in the setting of AMI and elevated PCWP appears multi-factorial. Although an increase in PCWP likely reflects increased LV volume (15), a number of factors may modify its degree and rate of rise. The PCWP may be greatly influenced by the functional capacity of the lymphatics as well as variations in interstitial, oncotic and hydrostatic pressure (16). The highly variable diastolic stiffness of the left ventricle and the impact of positive ventilation may also contribute to a heterogeneous response (17,18). The administration of vasopressor and vasodilator therapy and the difficulty in accurately examining the ventilated supine critically ill patient in the modern intensive care unit may also contribute to this observation.

It is unlikely that isolated RV infarction secondary to inferior myocardial infarction explains the findings observed in Group C. Patients diagnosed with CS secondary to RV

infarction were not considered for this analysis and are reported elsewhere (19). Only 5 of 82 (6%) patients with isolated hypoperfusion in Group C had a PCWP <12mm Hg. Further, mean right atrial pressure in Group C was similar to Group D. Left ventricular ejection fraction was similar and the PCWP was only marginally lower.

Readers should note that our patients differ from the classical description of isolated hypoperfusion resulting from RV involvement complicating inferior wall myocardial infarction (20). Patients with isolated hypoperfusion in these reports were chosen from the entire universe of AMI and were found to have normal to low PCWP and increased right atrial pressure secondary to hemodynamically significant RV infarction (20). By contrast, our patients with isolated hypoperfusion were selected for predominant LVF and have a mean PCWP of 22 ± 7 mm Hg and ejection fraction of 30%. Thus, although patients with hypotension secondary to RV infarction "respond in a hemodynamically beneficial manner to volume replacement" (20), the response to a volume bolus in our population with isolated hypoperfusion is uncertain and unreported. Although volume infusion may potentially harm some patients with very elevated PCWP, a potential benefit of moderate volume infusion for these patients cannot be ruled out, because volume administration may result in a rise in both PCWP and cardiac index. In the presence of a large infarction on surface ECG, echo documentation of RV and LV function are worthwhile before administering a significant fluid challenge.

The high mortality rates observed in Groups C and D are not surprising. Hemodynamic data, including cardiac output and PCWP were shown to be the strongest predictors of death in the Global Utilization of Streptokinase and tPA for Occluded Coronary Arteries (GUSTO) CS mortality model (21). Equally relevant is the fact that the physical findings of oliguria and cold peripheries were each independent and strongly predictive of mortality in this clinical setting (21, 22). These physical findings correlated more strongly with death than low BP. Both study Groups C and D have a low cardiac index in the setting of peripheral hypoperfusion—a harbinger of in-hospital death.

Previous studies. Elegant work performed in the past has validated the utility of the clinical examination in risk stratifying patients with AMI (20,23,24). Major limitations of the clinical prediction of the hemodynamic state have also been identified (20). These include the under-diagnosis of depressed cardiac index in 14% of patients and the failure to recognize an elevated wedge in about 15% of patients. The original report by Killip et al. (23) highlighted PC as a component of CS. Our experience shows that patients with clear lungs may be nevertheless in florid CS.

Study limitations. The use of RHC was not mandated by protocol, and the decision to place a RHC was local and investigator-dependent. Despite similar clinical findings, hemodynamics in those receiving and not receiving RHC may have been different. Although it is performed in tertiary critical care units, the accuracy of the measurements obtained with RHC cannot be verified by an independent investigator. The use of diuretic therapy prior to shock assessment was not recorded in the SHOCK Trial Registry, and the use of vasodilators (specifically, IV nitroglycerin) was not documented. These agents may have had a significant role in decreasing preload and mitigating PC. However the high PCWP and the similar right atrial pressures suggest that this did not significantly influence our findings. Patients enrolled in the SHOCK Trial Registry were not randomized to defined treatment strategies. Although the study groups appear adequately matched, a number of unrecorded variables may have influenced the subsequent outcome. There was no core laboratory for chest roentgenograms, and we acknowledge that the accuracy of physical findings is limited by clinical experience and subject to interobserver variability.

Conclusions. Right heart catheter utilization has declined in the setting of CS (25). The potential harm attributed to the RHC in the critical care setting may have contributed to this observation (26). This decline in invasive hemodynamic monitoring has important implications regarding the empiric administration of large intravenous fluid challenges to patients with large infarctions who present with hypotension and hypoperfusion. Non-invasive assessment of ventricular function is strongly advocated for patients with suspected CS.

Clinicians must be aware that PC may be absent when CS complicates AMI. Clinical hypoperfusion in the setting of AMI complicated by CS is a marker of in-hospital mortality irrespective of the presence or absence of PC. Early clinical recognition and triage of this group is clearly warranted and may have an impact on outcome.

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